Signaling and Communication in Plants

P. Vidhyasekaran

Switching on Plant Innate Immunity Signaling Systems

Bioengineering and Molecular Manipulation of PAMP-PIMP-PRR Signaling Complex



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Chapter 1 Introduction

Abstract Plant innate immune system is a surveillance system against possible attack by pathogens. It is quiescent in normal healthy plants. It is a sleeping giant and when awakened by specific signals it triggers expression of several defense genes. Unlike, transgenic plants developed by engineering disease resistance genes against specific pathogens, plants overexpressing the plant immune system awakened by the alarm signals PAMP and PIMP trigger expression of hundreds of defense genes conferring resistance against wide range of pathogens. Both PAMPs and PIMPs are perceived by plants as alarm signals by specific receptors called pattern recognition receptors (PRRs). PAMPs activate expression of the genes encoding various PRRs. Besides PAMP molecules, pathogens secrete another type of molecules called effectors. While the pathogen-derived PAMPs are involved in switching-on the plant immune responses, the effectors are involved in switchingoff the PAMP-triggered innate immunity. The effectors may also bind with PRRs and disrupt binding of PAMP with PRR in PAMP-PRR signaling complex to impede PAMP-triggered plant immunity. Effectors may bind with the PRR signal amplifier BAK1 and block the function of PAMP-PRR signaling complex. Early and robust activation of PAMP-PRR signaling complex before the pathogens invade and secrete virulence effectors seems to be necessary for triggering strong defense responses. Several PAMP formulations have been developed and foliar application of the formulations triggers the induction of plant immune responses. The time of application is very critical in enhancing the efficacy of the PAMPs in controlling diseases. The PAMPs should be applied prior to pathogen invasion. The concentration of the PAMP applied also determines the efficacy of the treatment in controlling diseases. Oligogalacturonates (OGAs), plant elicitor peptides (Peps), and PAMPinduced Peptides (PIPs) are the important PIMPs capable of switching on plant innate immune responses. Bioengineering technologies have been exploited to utilize PIMPs to develop transgenic plants expressing enhanced disease resistance. Bioengineering PRRs has been shown to be another potential technology to awaken the quiescent plant innate immunity for effective management of crop diseases. Intergeneric transfer of PRR has been achieved to develop disease-resistant crop plants. Transcription factors are the master switches, which regulate expression of defense genes in the PAMP-triggered plant immune signaling systems. Several transcription factors have been shown to trigger "priming" of defense responses and induce "Systemic Acquired resistance (SAR)" in plants. The plant defense activators benzothiadiazole, probenazole, tiadinil and ergosterol trigger the expression of

P. Vidhyasekaran, *Switching on Plant Innate Immunity Signaling Systems*, Signaling and Communication in Plants, DOI 10.1007/978-3-319-26118-8_1 transcription factors which modulate the expression of defense genes inducing disease resistance. The rhizobacterial strain *Pseudomonas fluorescens* WCS417r induces systemic resistance (ISR) by activating the transcription factor MYB72. The MYC2 transcription factor also has been shown to be involved in *P. fluorescens* WCS417r-induced priming to trigger ISR. Several WRKY, MYB, MYC, bZIP, EREBP, and NAC transcription factors have been engineered in various crop plants to develop disease resistant plants. Some transcription factors have been found to negatively regulate the expression of defense signaling systems. Silencing of the negative regulator transcription factors may be a useful strategy in developing disease-resistant plants. Several bioengineering and molecular manipulation technologies have been developed to switch on the 'sleeping' plant innate immune system, which has potential to detect and suppress the development of a wide range of plant pathogens in economically important crop plants. Enhancing disease resistance through altered regulation of plant immunity signaling systems would be durable and publicly acceptable.

1.1 Plant Innate Immunity Is a Sleeping Giant to Fight against Pathogens

Diseases caused by oomycete, fungal, bacterial, and viral pathogens cause enormous crop losses and in some areas the crop may be completely devastated (Vidhyasekaran 2004; Byamukama et al. 2015; Cohen et al. 2015; Han et al. 2015; Handiseni et al. 2015; Holmes et al. 2015; Sharma-Poudyal et al. 2015; Strehlow et al. 2015). Chemical control is widely practiced to manage fungal and oomycete pathogens (Gent et al. 2015; Handiseni et al. 2015). Frequent development of resistance to the modern fungicides in the field population of fungal/oomycete pathogens is a challenging problem in using the fungicides to manage these diseases (Miles et al. 2012; Gudmestad et al. 2013; Tymon and Johnson 2014; Fernández-Ortuňo et al. 2015; Hu et al. 2015; Keinath 2015; Saville et al. 2015; Zeng et al. 2015). Effective chemicals are still not available to control bacterial, viral, viroid, and phytoplasma diseases (Jones 2001; Vidhyasekaran 2004; Bradley 2008; Kanetis et al. 2008). Breeding varieties with built-in resistance may be the sound approach to manage diseases (Vidhyasekaran 2007; Singh et al. 2008; Tagle et al. 2015). However, new races of pathogens appear frequently and the resistance often breaks down (Sørensen et al. 2014; Kitner et al. 2015; Maccaferri et al. 2015). Breeding for quantitative resistance is useful (Yasuda et al. 2015), but it is difficult to achieve (Vidhyasekaran 2007; St Clair 2010; Zhang et al. 2015). Breeding for resistance against broad-spectrum of pathogens will be ideal, but the traditional breeding methods are inefficient (Vleeshouwers et al. 2008; Tran et al. 2015). An alternative technology based on switching on plant innate immunity using pathogen-associated molecular patterns (PAMPs) and pathogen-induced molecular patterns (PIMP)/ host-associated molecular patterns (HAMPs) has been developed recently for management of viral and bacterial diseases (Li et al. 2011; Pavli et al. 2011, 2012; Choi et al. 2012; Li et al. 2012), and also for management of a wide range of biotrophic, hemibiotrophic, and necrotrophic fungal and oomycete pathogens (Miao et al. 2010; Xu et al. 2010; Choi et al. 2012; Miao and Wang 2013).

Plant innate immune system is a surveillance system against possible attack by pathogens. It is quiescent in normal healthy plants. It is a sleeping giant and when awakened by specific signals it triggers expression of several defense genes. Several hundreds of defense genes have been detected in plants and these are involved in plant immunity (Coram and Pang 2005; Vega-Sánchez et al. 2005; Wang et al. 2005; Wilkinson et al. 2005; Hermosa et al. 2006; Vidhyasekaran 2007; Sun et al. 2008). These defense genes encode different pathogenesis-related proteins (Anand et al. 2003; Zhu et al. 2006; Sun et al. 2008) most of which inhibit growth of oomycete (Lee et al. 2000), fungal (Moravčikova et al. 2004; Pervieux et al. 2004; Chen et al. 2006; Zhu et al. 2006; Hernández-Blanco et al. 2007) and bacterial pathogens (Vidhyasekaran 2002). Several defense-related genes encode enzymes involved in biosynthesis of toxic compounds such as phytoalexins (Nawar and Kuti 2003; Liu et al. 2006; Chassot et al. 2008), phenylpropanoids and isoflavonoids (Farag et al. 2008), and terpenoids (Keeling et al. 2008), or enzymes involved in cell wallfortification (Hamiduzzaman et al. 2005; Flors et al. 2008). The triggered plant immune responses include accumulation of pathogenesis-related proteins, deposition of lignin and callose in the cell wall, and production of anti-microbial compounds (Tsuda and Katagiri 2010; Gimenez-Ibanez and Rathjen 2010). Plant innate immunity is a powerful weapon to fight against a wide range of plant pathogens. Plants have innate immunity system (Nicaise 2014; Vidhyasekaran 2014, 2015; Piasecka et al. 2015; Robinson and Bostock 2015; Schwessinger et al. 2015; Tena 2015) and this system provides basic protective functions against broadest range of pathogens (Boller and He 2009; Boutrot et al. 2010; Chen et al. 2010a, b; Dodds and Rathjen 2010; Park et al. 2010b; Shimizu et al. 2010; Segonzac and Zipfel 2011; Zamioudis and Peterse 2012; Li et al. 2014a, b; Vidhyasekaran 2014).

1.2 Potential Signals to Switch on Plant Immune System

The plant innate immune systems have high potential to fight against viral, bacterial, oomycete, and fungal pathogens and protect the crop plants against wide range of diseases (Knecht et al. 2010; Lacombe et al. 2010; D'Amelio et al. 2011; Hwang and Hwang 2011; Alkan et al. 2012). However, these plant immune systems are quiescent in healthy normal plants. Specific signals are needed to switch on the sleeping giant for exploiting the quiescent immune system for combating diseases. These signals are derived from invading pathogens and called 'pathogen-associated molecular patterns' (PAMPs). The immune system is activated on perception of the PAMP of invading pathogens (Nürnberger and Kufner 2011; Segonzac and Zipfel 2011). Potential pathogens contain several PAMPs and they serve as alarm signals to activate the plant innate immunity (Böhm et al. 2014; Vidhyasekaran 2014;

Zhang et al. 2014). PAMPs are detected not only in pathogens, but also in saprophytes, probably in all microbes. Hence the PAMPs are also called as microbeassociated molecular patterns (MAMPs) (Jeworutzki et al. 2010; Thomma et al. 2011). PAMPs/MAMPS are potential tools to activate plant immune systems and can be effectively used to manage crop diseases (Iriti et al. 2011; Choi et al. 2012; Dafermos et al. 2012; Li et al. 2012; Sanchez et al. 2012; Quang et al. 2015; Sathiyabama et al. 2014).

Besides the microbe-derived elicitors (MAMPs), some host plant-derived elicitors called pathogen-induced molecular patterns (PIMPs) or host-associated molecular patterns (HAMPs) have been shown to activate the plant innate immune system (Yamaguchi and Huffaker 2011; Vallarino and Osorio 2012; Bellincampi et al. 2014; Hou et al. 2014). The PIMPs/HAMPs (host-derived elicitors) function almost in the same fashion as the PAMPs function in switching on plant innate immunity (Denoux et al. 2008; Ferrari et al. 2013). PIMPs and PAMPs activate similar downstream responses using many of the same molecular components (Ryan et al. 2007; Krol et al. 2010; Postel et al. 2010; Qi et al. 2010; Yamaguchi et al. 2010; Huffaker et al. 2011). Both of them bind specific LRR receptors and both activate the same downstream signaling events (Yamaguchi et al. 2006; Huffaker and Ryan 2007; Krol et al. 2010).

Both PAMPs and PIMPs are perceived by plants as alarm signals by specific receptors called pattern recognition receptors (PRRs) (Nicaise et al. 2009; Brutus et al. 2010; Petutschnig et al. 2010; Shinya et al. 2010; Schulze et al. 2010; Segonzac and Zipfel 2011; Hann et al. 2014). Plants utilize the PRRs to recognize PAMPs/ MAMPs (Böhm et al. 2014; Macho and Zipfel 2014; Zhang et al. 2014). Most of the PRRs identified are receptor-like kinases (RLKs) and receptor-like proteins (RLPs) (Yang et al. 2012; Zhang et al. 2013; Zipfel 2014). PAMPs activate expression of the genes encoding various PRRs (Zipfel et al. 2004, 2006; Qutob et al. 2006; Lohmann et al. 2010). PRRs bind with PAMPs for their activation (Boutrot et al. 2010; Petutschnig et al. 2010). The PRRs recognize PAMPs and PIMPs and switch on the plant innate immunity (Mentlak et al. 2012).

1.3 Pathogens Possess Weapons to Switch-Off Plant Immune Systems

Besides PAMP molecules, pathogens secrete another type of molecules called effectors (Wu et al. 2011; Vleeshouwers and Oliver 2014). While the pathogen-derived PAMPs are involved in switching-on the plant immune responses, the effectors are involved in switching-off the PAMP-triggered innate immunity (Thomma et al. 2011; Wu et al. 2011; Cheng et al. 2012). The effectors secreted by various pathogens have been shown to suppress the PAMP-triggered immunity (Shan et al. 2008; de Jonge et al. 2010). Effectors induce susceptibility, mostly by suppressing PAMP-induced immune responses. The effector proteins target basic innate immunity in

plants (Boller and He 2009; Song and Yang 2010; Szczesny et al. 2010; Rajput et al. 2014; Zheng et al. 2014). Several bacterial pathogens use a specialized type III secretion system to deliver effector proteins into host cells to subvert PAMP-triggered host defense mechanisms, thereby promoting pathogenesis (Song and Yang 2010; Szczesny et al. 2010; Zhang et al. 2010; Wu et al. 2011; Akimoto-Tomiyama et al. 2012).

The effectors may also bind with PRRs and block plant defense responses in the plant cell (Xiang et al. 2008; Zeng et al. 2012; Rosli et al. 2013; Xin and He 2013). Effectors may disrupt binding of PAMP with PRR in PAMP-PRR signaling complex to impede PAMP-triggered plant immunity (Mentlak et al. 2012). Some effectors have been shown to degrade the PRRs through ubiquitin-proteasome pathway and inhibit PAMP-triggered immunity (Göhre et al. 2008; Gimenez-Ibanez et al. 2009a, b). Effectors may also target the kinase domains of PRR and inhibit the PRR receptor kinase activity to block PAMP-triggered immunity (Shan et al. 2008; Xiang et al. 2008; Zipfel and Rathjen 2008; Xiang et al. 2011). The effectors may also inhibit the autophosphorylation of PRRs to suppress the PAMP-triggered immune system (Xiang et al. 2008). The effectors may prevent the activation of PRR signaling complex by inhibiting the autophosphorylation of PRRs (Hann and Rathjen 2007). Effectors may bind with the PRR signal amplifier BAK1 and block the function of PAMP-PRR signaling complex (Shan et al. 2008; Xiang et al. 2008; Hann et al. 2008; Hann et al. 2010).

1.4 Bioengineering and Molecular Manipulation Technologies to Switch on the Sleeping Quiescent Plant Immune System to Win the War against Pathogens

Several bioengineering and molecular manipulation technologies have been developed for management of a broad-spectrum of diseases caused by a wide-range of viral, bacterial, fungal and oomycete pathogens, exploiting the potential of plant innate immunity (Ferrari et al. 2008; Lacombe et al. 2010; Hwang and Hwang 2011; Volpi et al. 2011; Alkan et al. 2012; Ferrari et al. 2012; Li et al. 2012; Wang et al. 2013; Fu et al. 2014; Lloyd et al. 2014; Macho and Zipfel 2014; Trouvelot et al. 2014). The crop diseases can be controlled by switching on plant innate immunity by manipulating PAMP-PIMP-PRR signaling complex. Early, rapid and strong activation of plant innate immune system is necessary to induce strong defense responses against pathogens. Early and robust activation of PAMP-PRR signaling complex before the pathogens invade and secrete virulence effectors seems to be necessary for triggering strong defense responses and for effective management of crop diseases (Orlowska et al. 2011; Aghnoum and Niks 2012; Lanubile et al. 2012).

Several PAMP formulations have been developed and foliar application of the formulations triggered the induction of plant immune responses (Dong et al. 2004;

Elmer and Reglinski 2006; de Capdeville et al. 2008; Shao et al. 2008; Iriti et al. 2011; Dafermos et al. 2012; Chuang et al. 2014). Several factors such as environment, genotype, and crop nutrition determine the efficacy of the PAMPs in controlling diseases under field conditions (Walters et al. 2005). The time of application is very critical in enhancing the efficacy of the PAMPs in controlling diseases (de Capdeville et al. 2002; Agostini et al. 2003). The PAMPs should be applied 2–5 days prior to pathogen invasion (Qiu et al. 2001; de Capdeville et al. 2002, 2003). The concentration of the PAMP applied also determines the efficacy of the treatment in controlling diseases (de Capdeville et al. 2002; Chen et al. 2008). Variability in structure and function has been reported among various PAMPs (Che et al. 2000; Tanaka et al. 2003; Fujiwara et al. 2004). The time of induction (Luna et al. 2011), intensity of induction (Lecourieux et al. 2002, 2005), and duration of induction (Aziz et al. 2007) of the defense signals may vary depending on the type of PAMPs. Efficacy of PAMPs in controlling diseases may also vary depending on the challenging pathogens (Agostini et al. 2003). Hence, suitable PAMPs have to be selected for management of various crop diseases.

Bioengineering PAMP genes has been shown to be powerful tool to trigger plant immune responses (Keller et al. 1999; Li and Fan 1999; Belbahri et al. 2001; Choi et al. 2004; Donghua et al. 2004; Peng et al. 2004; Takakura et al. 2004, 2008; Malnoy et al. 2005; Jang et al. 2006; Ren et al. 2006a, b; Cai et al. 2007; Sohn et al. 2007; Shao et al. 2008; Qiu et al. 2009; Huo et al. 2010; Miao et al. 2010; Xu et al. 2010; Pavli et al. 2011, 2012; Choi et al. 2012; Li et al. 2012; Miao and Wang 2013; Ouang et al. 2015). Levels of PAMP gene expression may vary among different transgenic plant lines developed by bioengineering technologies (Peng et al. 2004). The line, which shows high level of PAMP gene expression, shows very high level of resistance against pathogens, while the line, which shows low level of expression of the PAMP gene shows only low level of resistance (Peng et al. 2004). Hence, the transgenic lines should be carefully selected to generate highly useful diseaseresistant cultivars. Expression of PAMP genes can be enhanced by properly selecting the promoter for gene transcription (Takakura et al. 2004). The transgenic plants expressing introduced gene may have side effects, showing retardation of plant growth and reduced crop yield potential. However, transgenic plants expressing the PAMP harpin gene show good agronomic characters (Xu et al. 2010; Li et al. 2011; Pavli et al. 2011). Selection of suitable pathogen-inducible promoter for expressing the PAMP gene appears to be a perquisite for developing disease-resistant plants without any reduction in yield potential (Choi et al. 2004; Donghua et al. 2004)

Bioengineering technologies have been exploited to utilize PIMPs/HAMPs to develop transgenic plants expressing enhanced disease resistance. Oligogalacturonides are the best-characterized plant cell wall-derived PIMPs/ HAMPs (Vallarino and Osorio 2012). However, not all OGAs are capable of elicit-ing a defense response. Their ability to elicit defense responses depends on length (degree of polymerization), degree of methyl esterification and the level of acetylation (Côté and Hahn 1994; Vidhyasekaran 1997, 2007; Wiethölter et al. 2003; Aziz et al. 2004; Ferrari et al. 2007; Osorio et al. 2008; Vallarino and Osorio 2012).

Both the degrees of substitution (methylesterification and/or acetylation) and polymerization can be controlled by specific enzymes such as pectin methylesterases (PMEs), pectin acetylesterases (PAEs), polygalacturonases (PGs), or pectate lyases-like (PLLs) (Sénéchal et al. 2014). PME can modify the structure of OGAs and the modified OGAs will be highly active in triggering plant innate immune signaling systems. Transgenic plants expressing pectin methyl esterase gene (*PME*) generate oligogalacturonides, which act as host-derived elicitor/PIMP/HAMP. These transgenic plants show enhanced expression of plant immune responses and enhanced disease resistance (Lionetti et al. 2007, 2014; Osorio et al. 2008, 2011). PME activity is tightly regulated by an inhibitor protein called pectin methylesterase inhibitor protein (PMEI) (Giovane et al. 2004; Di Matteo et al. 2005). Transgenic plants overexpressing genes encoding PME inhibitor proteins show enhanced disease resistance (Lionetti et al. 2007, 2014; An et al 2008).

Transgenic plants expressing PG gene show enhanced disease resistance (Ferrari et al. 2008). Polygalacturonase-inhibiting proteins (PGIPs) play important role in switching on plant immune signaling systems (Manfredini et al. 2005; Federici et al. 2006; Alexandersson et al. 2011). Transgenic plants expressing *PGIP* genes also show enhanced disease resistance (Joubert et al. 2006, 2007; Alexandersson et al. 2012; Nguema-Ona et al. 2013; Wang et al. 2013). The expression of *PGIP* genes does not affect the agronomic characters of the transformed plants (Powell et al. 2000; Capodicasa et al. 2004; Agüero et al. 2005; Borras-Hidalgo et al. 2012; Nguema-Ona et al. 2004; Agüero et al. 2005;

Plant elicitor peptides (Peps) are the other group of PIMPs/HAMPs (Huffaker et al. 2011; Logemann et al. 2013; Hann et al. 2014). The Pep proteins are processed from the precursor PROPEP proteins. Transgenic plants overexpressing *PROPEP* genes show enhanced disease resistance (Huffaker et al. 2006). Transgenic plants overexpressing PIP (PAMP-induced Peptides) and systemin switch on plant innate immunity and show enhanced disease resistance (Coppola et al. 2014; Hou et al. 2014). PIMPs/HAMPs appear to be powerful tools to engineer disease resistance in field crops.

Bioengineering PRRs has been shown to be another potential technology to awaken the quiescent plant innate immunity for effective management of crop diseases. Intergeneric transfer of PRR from the weed plant Arabidopsis to various crop species has been achieved to develop disease-resistant crop plants. EFR is a Brassicaceae-specific PRR (Zipfel et al. 2006). Transfer of EFR from *Arabidopsis* to various crop plants is highly useful for crop disease management. Pathogens that are adapted to a particular host plant may be adept at suppressing the PRRs of that host by their effectors. The effectors of the pathogens might not recognize PRRs from other host plants and development of transgenic plants expressing PRRs from other plant species may provide good resistance against various bacterial pathogens possessing the PAMP EF-Tu (Lacombe et al. 2010). Transgenic tomato plants expressing *EFR* gene from *Arabidopsis* show enhanced resistance against the tomato wilt pathogen *Ralstonia solanacearum* (Lacombe et al. 2010). Transgenic tobacco expressing EFR also show resistance against *Agrobacterium. tumefaciens* (Brutus et al. 2010). Transgenic banana plants expressing the rice PRR *XA21* gene

showed complete resistance to *Xanthomonas campestris* pv. *musacearum* (Tripathi et al. 2014). Transgenic *Citrus sinensis* plants expressing the rice PRR *XA21* gene enhance resistance against the citrus canker pathogen *Xanthomonas axonopodis* pv. *citri* (Mendes et al. 2010). Transgenic rice plants overexpressing *Xa21* gene showed enhanced resistance against the rice bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* (Park et al. 2008, 2010a, b; Chen et al. 2014). Transgenic *Arabidopsis* plants expressing *FLS2* gene showed increased resistance against *Pseudomonas syringae* pv. *tomato* DC3000 (De Lorenzo et al. 2011). The transgenic rice plants overexpressing the HAMP receptor *WAK1* show enhanced disease resistance (Li et al. 2009).

Transcription factors are the master switches, which regulate expression of defense genes in the PAMP-triggered plant immune signaling systems (Century et al. 2008; Moreau et al. 2012). PAMPs and PIMPs/HAMPs switch on the expression of various transcription factor genes involved in plant defense responses (Denoux et al. 2008; Higashi et al. 2008; Chujo et al. 2013; McLellan et al. 2013). Several transcription factors have been shown to trigger "priming" of defense responses and induce "Systemic Acquired resistance (SAR)" in plants (Chavan and Kamble 2013; Nakayama et al. 2013). The plant defense activators benzothiadiazole, probenazole, and tiadinil trigger the expression of transcription factors which modulate the expression of defense genes inducing disease resistance (Shimono et al. 2007, 2012). DL-3-aminobutyric acid (β-aminobutyric acid, BABA) has been found to induce priming of WRKY transcription factors and trigger systemic resistance (Jakab et al. 2001). Foliar spray with BABA led to a significant reduction of lesion development in Brassica carinata caused by Alternaria brassicae (Chavan and Kamble 2013). Ergosterol treatment triggered a 23-fold increase of VvWRKY gene expression in grape plantlets and induced resistance against the necrotrophic fungal pathogen Botrytis cinerea (Laquitaine et al. 2006). The rhizobacterial strain Pseudomonas fluorescens WCS417r induces systemic resistance (ISR) in A. thaliana by activating the transcription factor MYB72 (Van der Ent et al. 2008). The MYC2 transcription factor also has been shown to be involved in P. fluorescens WCS417r-induced priming to trigger ISR (Pozo et al. 2008).

Several WRKY, MYB, MYC, bZIP, EREBP, and NAC transcription factors, have been engineered in various crop plants to develop disease resistant plants (He et al. 2001; Shin et al. 2002; Fischer and Dröge-Laser 2004; Guo et al. 2004; Cao et al. 2006; Waller et al. 2006; Chujo et al. 2007; Kim et al. 2007; Marchive et al. 2007; Mid et al. 2007; Qiu et al. 2007; Wang et al. 2007; Zuo et al. 2007; Dai et al. 2008; Zhang et al. 2008; Bahrini et al. 2011a, b; Fan et al. 2011; Abbruscato et al. 2012; Liu et al. 2012; Peng et al. 2012; Shimono et al. 2012; Yu et al. 2012; Zhu et al. 2012; Han et al. 2013; Lee et al. 2013; Marchive et al. 2013; Wei et al. 2013; Chujo et al. 2014; Dang et al. 2014; Yokotani et al. 2014; Cao et al. 2016; Cheng et al. 2015; Jisha et al. 2015; Li et al. 2015; Shan et al. 2015).

Most of the successful stories in management of crop diseases using transcription factors are in rice plants. Transgenic rice plants overexpressing *OsWRKY13* showed enhanced blast (*Magnaporthe oryzae*) and bacterial blight (*Xanthomonas oryzae* pv. *oryzae*) disease resistance (Qiu et al. 2007; Cheng et al. 2015). WRKY30overexpressing rice plants show resistance against the rice blast pathogen *M. oryzae*, the sheath blight pathogen *Rhizoctonia solani* and the bacterial blight pathogen *X. oryzae* pv. *oryzae* (Peng et al. 2012; Han et al. 2013; Lee et al. 2013) Transgenic rice plants overexpressing *OsWRKY31* (Zhang et al. 2008), *WRKY45* (Shimono et al. 2007, 2012; Goto et al. 2015), *OsWRKY47* (Wei et al. 2013), *WRKY53* (Chujo et al. 2007, 2014), OsWRKY89 (Wei et al. 2013) show enhanced resistance to *M. oryzae*. Transgenic rice plants overexpressing *OsWRKY71* showed enhanced resistance to the bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* (Liu et al. 2007).

Some transcription factors have been found to negatively regulate the expression of defense signaling systems. Silencing of the negative regulator transcription factors may be a useful strategy in developing disease-resistant plants. The *TaNAC1* gene-silenced wheat plants showed enhanced resistance against the stripe rust pathogen (Wang et al. 2015). *WRKY42*-suppressing (*WRKY42*-RNA interference [RNAi]) rice plants were developed and these plants showed increased resistance to *M. oryzae* (Cheng et al. 2015).

1.5 Switching on Plant Innate Immunity Using PAMP-PIMP-PRR-Transcription Factor Is the Most Potential Biotechnological Approach for Management of Crop Diseases

Susceptibility and resistance are two sides of the same coin (Vidhyasekaran 2007). The plant immune system is induced faster and to a higher level in resistant interactions (Makandar et al. 2006). The major differences between susceptible and resistant interactions are the magnitude and timing of induction of plant immune signaling system (Makandar et al. 2006; Rinaldi et al. 2007; Asselbergh et al. 2008). Higher and faster expression of genes involved in signal transduction systems has been found to be associated with improved tolerance to pathogens (Coppinger et al. 2004; Waller et al. 2006; Yamamizo et al. 2006; Zhang et al. 2006; Brader et al. 2007; Qiu et al. 2007). PAMP-induced defense in susceptible host plants is insufficient to stop infection; nonetheless, it is referred to as basal resistance (Nürnberger and Lipka 2005; Fung et al. 2008). Early, rapid and strong activation of plant innate immune system is necessary to induce strong defense responses against pathogens (Orlowska et al. 2011; Aghnoum and Niks 2012; Lanubile et al. 2010, 2012, 2014; Groβkinsky et al. 2012). Strong signals are needed to switch on early and strong activation of plant immunity. Engineering and/or proper application of PAMP/PRR products much before pathogen invasion results in early switching on the plant immune system.

Recently several bioengineering and molecular manipulation technologies have been developed to switch on the 'sleeping' plant innate immune system, which has potential to detect and suppress the development of a wide range of plant pathogens in economically important crop plants (Lacombe et al. 2010). Enhancing disease resistance through altered regulation of plant immunity signaling systems would be durable and publicly acceptable (Yamamizo et al. 2006; Shao et al. 2008; Gust et al. 2010; Lacombe et al. 2010). The plant innate immune systems have high potential to fight against viral, bacterial, oomycete, and fungal pathogens and protect the crop plants against wide range of diseases (Knecht et al. 2010; Lacombe et al. 2011; Hwang and Hwang 2011; Alkan et al. 2012). This book describes various bioengineering and molecular manipulation technologies employed to trigger defense responses and manage crop diseases.

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